A TELEVISION RADIOGRAPHIC EVALUATION OF THE ASSOCIATION BETWEEN DENTIN SCLEROSIS AND PULPAL FLOOR WIDTH

by

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"We find in research that a certain amount of intelligent ignorance is essential to human progress, because if you know too much you won't try the thing."

Kettering

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INTRODUCTION

Dentists, for many years, regarded dentin as a hard tissue that was cut mainly for restorative procedures, not as a part of a vital tissue that reacted to injury. Reactions by the tooth in response to injury and cavity preparation have been limited mostly to the effects on the dental pulp. However, a growing awareness of the ability of dentin to react to a stimulus is evidenced by a review of the dental literature.

The instrumentation of primary teeth during cavity preparation, on many occasions closely approximates pulpal tissue. The dentin, exposed by caries may be an ineffectual barrier in preventing a pulp reaction. One should, therefore, take full advantage of the inherent properties of dentin in establishing a barrier resulting from its altered reactivity. In recent years, a base of calcium hydroxide and methyl cellulose has been advocated. Investigators[1,2] observed concomitant radiographic changes under restorations using this base material. Indeed, the calcium hydroxide methyl cellulose acted as a "trigger mechanism" in initiating the dentin to react to this specific stimulus. The result was the increased deposition of hypercalcified dentin.

In view of these observations, the question arose as to whether an association existed between the depth of the carious lesion and the formation of dentin sclerosis deposited beneath the restoration lined with calcium hydroxide. Television densitometric instrumentation was employed to electronically measure these changes on pre and postoperative serial radiographs of teeth with deep carious lesions that were restored with a calcium hydroxide and methyl cellulose base material.

REVIEW OF THE LITERATURE

Physiology of Dentinal Sclerosis

Dentin Sclerosis, a reaction to the carious process, abrasion, or cavity preparation, has been referred to as a protective mechanism inherent in human tooth structure. Beust[3] emphasized the importance of this protective mechanism by stating that the dental caries resistance of a tooth increases in proportion to the amount of its dentin sclerosis. This mechanism of dentin sclerosis has been an area of considerable controversy and has been reviewed by numerous authors in the dental literature.

Hunter[4] in 1839 was the first investigator to notice that "as a result of abrasion and in the face of possible pulp exposure, the bottom part of the cavity is filled with a new material." John Tomes[5] described a similar area in dentin with calcified fibrils and a greater power of resisting decomposition than surrounding dentin. Tomes called this area of hypercalcification "translucent dentin." Charles Tomes[6] referred to sclerotic dentin as a decalcification, in contrast to the original views of Sir John Tomes, who attributed it to a hypercalcification. The variation in opinions arose because the former investigator was describing the decalcified zone of carious dentin which lies in close proximity to the sclerotic dentin zone being described by the latter investigator. Salter[7] in 1865 noted the filling of the dentinal tubules with a secondary deposit, a condition he called "horny dentin" with the tissue being more or less clear because of tubule obliteration and subsequent light refraction. Inglis[8] in 1900 stated that a recalcification of dentin would be advantageous in protecting the pulp against thermal irritation from without. Beust,[9] on examination of the dentinal tubules of young erupted teeth, noted a consistent uniformity in the translucency of the individual tubules, a condition to which he also ascribed the term "translucent dentin." He further concluded that this modification was a normal biological process supporting the views of Mummery,[6] Lefkowitz,[10] and Vissotzsky[11] that the vitality of the primary dentin is important in the production of the tissue changes.

Tissue changes in primary dentin are described in a varied and confusing manner. Terminology such as transparent dentin, opaque dentin, hyalin dentin, as well as horny dentin and metamorphosed dentin are employed interchangeably throughout the literature. Mjor[12] believes that the possibility exists that these terms reflect the same basic change and the variation is descriptive of different stages of the same process.

A great variance of opinion exists as to the physiological process which culminates in the formation of sclerotic dentin. Bodecker[13] stated that the changes which take place beneath a carious lesion were the results of a "fatty metamorphosis" of the dentinal fibrils. Often as a result of abrasion of the occlusal surfaces of teeth, secondary dentin is laid down in the pulp chamber between the odontoblasts and the previously formed dentin. This process cuts off the dentinal fibrils from their vital connections, the odontoblasts, resulting in the fatty metamorphosis.

Hatton[14] also recognized a "fatty metamorphosis." He reported that the metabolic activities in the tubules were interfered with, resulting in the demonstration of free fat in the tubules and supposedly within Tomes' processes as a result of caries, abrasion or erosion. Free fat, in contrast, was not found in these tubules as long as they were not damaged. According to Hatton, lime salts are deposited or precipitated in the dead organic matrix filling the tubule, resulting in the tubular contents becoming sclerosed. This takes place at a later stage in the degenerative process, again presumably because of a change in the metabolism and probably because of the accumulation of a matrix of non-vital organic matter in the tubule.

In 1932, Beust[15] published a critical review of Bodecker's theory of caries resistance. He questioned the feasibility of the "fatty metamorphosis" postulate as described by Bodecker. Beust conducted an experiment in order to determine whether fat contributed to this condition. Eight teeth were halved longitudinally in such a manner as to expose the pulp chambers. These were then passed at 24 hour intervals through liberal amounts of graded alcohols followed by absolute alcohols. They were then transferred to fresh absolute alcohol for three days. This was followed by a bath of anhydrous ether, and a return to alcohol and basic fuchsin solution for 10 days. It was inferred that through this treatment any fat occluding the tubules would have been removed and their permeability restored. The results showed that tubule blockage was still evident, disproving the theory of Bodecker. Beust concluded by stating "that the theory as propounded by Bodecker in 1929 embodied conceptions worthy of consideration by those who are convinced of the occurrence of metabolic changes in enamel. In the light of the investigations into tooth maturation by the writer, however, which show that the lymph flow diminishes with the increase of resistance to caries, the theory has lost its major promise." Beust maintained that opaque areas in the dentin were primarily due to variation in calcification of these tissues.

Siegmund and Weber[16] attributed the deposition of visible fat globules observed in caries to injury of the cells by toxins or other irritants. They assumed that visible fat was not necessarily pathologic, pointing out that the presence of fat in teeth was a normal ingredient of the dentin at all ages. However, paradoxical to Bodecker's theory, they also observed less fat in the teeth of the aged (whose immunity to caries is most pronounced) than in the teeth of the young.

Fish[17] believed that calcification was initiated by the activity of the odontoblastic fibrils becoming sclerosed with no fibril necrosis or death occurring. Black[18] stated that abrasion of tooth structure results in death of dentinal tubules with a definite loss of dentin strength. However, investigators such as Scott and Symons[19] suggest that the intensity of the stimulus is the major factor. Dentinal sclerosis forms in response to mild stimulation. Increased stimulation, in contrast, results in the destruction of the odontoblastic process, and formation of a dead tract.

Bradford[20] considers the odontoblast cell as amoeboid-like, with the body of the cell in the pulp and a long pseudopodium reaching out through the dentin to the amelo-dentin junction. Mild stimulation of the pseudopodium will cause its gradual withdrawl into the body of the cell. As it is withdrawn, the cell lays down material to occlude the tubule and subsequent calcification produces sclerosis. Under slightly increased stimuli, the pseudopodium may be withdrawn hastily and the material deposited may not be sufficiently organized to calcify so that the dentin will appear as a "dead tract," originally described by Fish.[21] Bradford also stated "that both sclerosis and dead tracts are reactions of dentin to stimuli, and the type of response elicited depends upon three factors: (1) Size of tubule - small size tubules will form sclerotic dentin in contrast to large tubules which tend to form dead tracts; (2) Vitality of the odontoblast - the younger odontoblast will be more capable of forming sclerotic dentin more readily, thereby verifying the postulate of Lefkowitz[22] that 'dead tract formation is more usual in the teeth of older people and sclerosis in younger people.' (3) Initial size of the tubule - since the initial dentin tubule is quite large, more dead tract formation might be expected in the very young in contrast to the adolescent. This lack of sclerosis should reduce the resistance of the dentin to the advancing carious lesion. Clinically, it is common knowledge that caries which occur in recently erupted teeth progress much more rapidly than in teeth which have been in the oral cavity for a number of years, and the degree of sclerosis may be the prime factor." Bradford postulated that if sclerosis of the dentin is complete, caries will only be able to penetrate the tissue very slowly by a "gradual proteolysis of the inter-tubular dentin matrix." Under these circumstances, bacterial invasion of the tissues will be a very late stage in the disease process and the clinical carious lesion will be very slow to develop. The lesion will tend to spread along the plane of least resistance (the amelodentinal junction) rather than towards the pulp, resulting in undermined enamel and subsequent saucer-shaped lesions. This characteristic pattern is frequent in neglected mouths where the reaction of dentin has been favorable. Conversely, if the degree of sclerosis is slight the bacterial invasion of the tissues will occur as soon as a pathway through the enamel has developed. In such cases, carious involvement of the pulp will ensue rather rapidly.

Blake[23] concluded that the translucent and impermeable regions that develop in both the crown and root are shown to be associated with the closure of dentinal tubules with a mineral substance of high radiopacity. In affected regions all stages of narrowing during closure of the tubules can be found, supporting the view that this is a reaction of individual odontoblastic dentinal fibril systems and not a general deposition of calcified material throughout the dentin. Blake stated that the process of sclerosis was probably a continuation of the normal peritubular calcified sheath formation. Like the normal peritubular zone, this material occluding the tubules is more radiopaque than the remaining intertubular dentin, and on decalcification leaves no organic matrix that can be demonstrated by normal histological methods so that the tubules which have been occluded are not distinguishable in decalcified preparations. Harcourt,[24] in describing the peritubular zone, stated that it varies according to its position, the age of the tooth, and whether or not the tooth has been injured by caries. Van Huysen,[25] however, used an unerupted human premolar tooth to demonstrate, by utilizing historadiographs and photomicrographs, that the peritubular calcification of the major portion of the dentin of a young tooth is quite normal and cannot be attributed to a reaction to injury, attrition, age changes or dental caries. In another study, Van Huysen[26] compared x-ray micrographs of normal and sclerosed dentin. Planoparalled histological sections 30 microns thick were prepared by grinding undecalcified dentin from an unerupted first premolar and an exfoliated deciduous tooth crown which showed dentin sclerosis between the filling and the pulp chamber shadow on the clinical radiograph. He concluded that the dentin develops peritubular calcification shortly after its formation, and under stimuli from outside irritation sclerosis of dentin extends from the peritubular calcification areas peripherally out into the intertubular matrix; thus tubular obliteration may occur without sclerosis.

This finding is in agreement with Mjor[12] who suggests the term "secondary intradentinal mineralization" to signify a general increase in mineralization without specifically limiting the change to alterations in the dentinal tubules. Apparently obliteration of the tubules with mineral salts takes place from the periphery of the tubule, that is from the dentinoenamel margin, and progresses centripetally along the tubule towards the pulp chamber. According to Harcourt[24] the sclerotic zone is narrowest at the pulp and widest at the dentino-enamel junction. In longitudinal sections, this gives the appearance of a more radiopaque strip overlying the affected dentin, while in transverse sections, it appears as a ring or ellipse of tubules blocked with hypermineralized plugs.

Physical Properties of Dentinal Sclerosis

A number of reports have been published describing the properties of dentin sclerosis. Bergman and Engfeldt[27] used light photomicrographs and historadiographs to show dentinal tubules in a cross-section derived from a carious tooth. They believed that the bright rings about the dentinal tubules represented peritubular calcification. Many of the tubules were completely x-ray opaque, as though obliterated by mineral salts. It was the opinion of these investigators that the x-ray translucent tubules with bright, x-ray opaque rings surrounding represented forms transitional between unaffected and obliterated tubules initiated by adjacent caries. Rockert[28] used x-ray microradiographs and indicated that the peritubular x-ray opacities represented increasing obliteration of dentinal tubules. He correlated this observation with an increase in age of the individuals from whom the teeth were taken. Dreyfuss, Frank, and Gutmann[29] observed that microradiographic aspects of dentinal sclerosis were identical regardless of the origin being abrasion, attrition, caries, or oldness. Van Huysen[26] indicated that the x-ray opaque or obliterated dentinal tubules were also more numerous in the peripheral dentin than near the pulp. Thus, peritubular hypercalcification, according to Van Huysen, probably commences throughout the dentin soon after its formation, but tubule obliteration takes place from the periphery toward the pulp chamber.

Little is known about the true nature of the organic matrix constituting the peritubular zone. Frank[30] stated that it may be similar to the intertubular substance, differing only in that it may be impregnated with a larger amount of apatite crystals. However, Nalbandian et al,[31] using optical electron and x-ray microscopy, observed that sclerosed tubules appeared to have a different texture and consistency from that of intertubular dentin. Mjor,[32] in contrast, using a ground section of a non-carious unoperated molar, stated that no appreciable difference existed between the calcified tissue lining the dentinal tubules and that found in the intertubular areas.

Hardness studies by investigators such as Bradford[33] revealed that sclerotic dentin is harder than normal dentin. Bradford described the area of sclerosis as being hard, as well as being "crystalline and brittle." Hodge and Mc Kay[34] reported a definite increase in microhardness of dentin below the zone of active caries.

Permeability studies conducted by Beust[3] and Fish[17] indicated that the dentinal tubules, modified by slowly progressing caries, were impermeable to dyes. Fish utilized a methyl blue dye to demonstrate the impervious nature of affected tubules. Miller[35] stated that increased resistance to acids and dyes was unique to sclerotic dentin. Van Huysen[36] considered the increased opacities a result of additional amounts of material, probably calcific, offering an increased resistance to the passage of roentgen rays.

The degree of change in opacity differs in various teeth, depending on the structure of the tooth, the activity of the irritant, or both. Van Huysen[37] reported that transparent dentin absorbs from three to four percent more roentgen radiation than unmodified dentin. In another study by Van Huysen et al,[38] the investigators used a standard ionization chamber method in addition to a densitometric method for measuring roentgen-ray absorption. The dentin studied was obtained from a sample of 73 teeth. Data showed (1) different teeth from the same or different mouths have different absorption values for a given thickness of dentin unaffected by caries; (2) areas of dentin affected by dental caries give increases in roentgen-ray absorption of five to 40 percent when compared with normal dentin of the same tooth; (3) marked increases in the roentgen-ray absorption of dentin affected by dental caries, ie: 10 to 40 percent, are the exception rather than the rule.

Cape and Kitchen[39] described a peculiar luminous birefringence which they thought was a result of the removal of the inorganic substance from the particular area, or a marked increase in the birefringence of the organic material. The investigators state that this increase in birefringence may function as a part of the defense mechanism against caries.

The Relationship of Calcium Hydroxide To Dentinal Sclerosis

The use of calcium hydroxide as a pulp capping agent has received a great deal of attention from dental investigators. The first recorded use of calcium hydroxide was by Hermann[40] in 1930. He introduced calcium hydroxide in the form of a paste, Calxyl, containing calcium hydroxide, sodium chloride, potassium chloride, calcium chloride and sodium bicarbonate. Hermann described the appearance of a dentinal bridge when Calxyl was used as a dressing in pulpotomies.

Calcium hydroxide was first introduced in the United States in 1930 by Teuscher and Zander.[41] These investigators capped exposed pulps after a pulpotomy procedure with a mixture of Calxyl. Histological examination after three and one-half months on one primary and one permanent tooth revealed complete bridge formation with the underlying pulp free of inflammation.

Zander[42] in 1939 postulated on the possible action of calcium hydroxide when in contact with vital pulp tissue. He stated that "The blood is normally saturated or supersaturated with calcium and phosphate ions and, hence any increase in the calcium and phosphate ions would cause a precipitation or laying down of calcium salts. A material which contains either calcium or phosphate in a combination which would be easily ionized when brought in contact with the surface of the pulp should react in this manner. This probably is the action of calcium hydroxide. The normal mechanism of deposition of bone salts has been shown to depend to a large extent on an increase of phosphate ions due to the liberation of inorganic phosphate from blood or tissues by means of a phosphatase enzyme. As bone phosphatase is known to act best in an alkaline medium and as the solubility product decreases with increased alkalinity the conditions here approach an optimum. Calcium hydroxide has a pH of 12.4."

Berk[43] in 1950 was the first investigator to incorporate aqueous methyl cellulose as a vehicle in calcium hydroxide. Berk felt that the combination of calcium hydroxide methyl cellulose as a paste simplified its mode of application because of its smooth texture and cohesive properties. Six dogs' teeth were mechanically exposed and capped with the calcium hydroxide methyl cellulose paste. Histological sections taken at two and one-half months revealed healing pulps in all of the teeth, together with the formation of new odontoblasts and a covering of dentin.

In 1958, Klein[1] conducted a densitometric study to determine whether a relationship existed between deciduous dentin sclerosis and calcium hydroxide methyl cellulose base material. Complete caries removal was conducted on

351 deciduous teeth. A total of 191 of the teeth were restored with a calcium hydroxide methyl cellulose base material followed by an amalgam restoration. The remaining 160 teeth did not receive the base material. Periodic radiographs were taken and in 93 percent of the teeth treated with the base material a characteristic transparent white area of sclerosis was observed beneath the restoration. In contrast, 99 percent of the 160 teeth which did not receive the base material, failed to demonstrate dentin sclerosis formation.

Mjor[2] in 1960 conducted a study to determine the changes in mineralization of dentin produced by calcium hydroxide and amalgam. Dentin of 25 human, non-carious young vital teeth exposed by cavity preparation and covered with a calcium hydroxide and water mixture showed a marked increase in mineralization (secondary intradental mineralization). Mjor concluded that the vitality of the tooth appeared to be of great importance in producing tissue changes in dentin. A less marked increase in mineralization was found in calcium hydroxide covered dentin of extracted teeth. The area of increased mineralization was found between the calcium hydroxide and the predentin. It was limited in extent to include only the portion of dentin permeated by the tubules exposed by cavity preparation and covered by calcium hydroxide.

Mjor, Finn and Quigley,[44] in another publication, concluded that the effect of the length of time the calcium hydroxide covered the dentin was not clear. However, they observed changes in mineralization occurring during short periods of time, some within 15 days. They also observed that the difference in hardness did not correlate with cavity preparation depth.

Seltzer and Bender[45] stated that "the application of calcium hydroxide to dentin causes sclerosis of the primary tubules but does not stimulate the laying down of reparative dentin."

Dentinal Sclerosis Concept Controversy: A Possible Mode of Action

Klein[46] in conducting a historadiographic study on deciduous teeth lined with calcium hydroxide noted a rythmic calcification in the radiographically evident areas of sclerosis. These zones appeared to radiate out from the pulpal horn and were classified as follows:

1. Zone of Matrix Hypercalcification - where the dentinal matrix is hypercalcified.

2. Zone of Matrix Moderate Calcification - an area where the dentinal matrix is moderately calcified.

3. Zone of Tubule Obliteration - an area in which a band of dentinal tubules appears completely obliterated by hypercalcified matrix.

He described a possible chain of events that may be the method of hypercalcification in primary dentin.

Densities of the peritubular rings were twice the density of the matrix in the zone of hypercalcified matrix. The densities of both the matrix and peritubular rings in the zone of moderate calcification were about one-half of the density of the hypercalcified matrix. Readings in the zone of obliterated tubules were nearly the same as those in the zone of moderate matrix calcification although a definite band of obliterated tubules was noted. Klein theorized that the dentinal tubules acted as a nidus for an increase in calcification of the dentinal matrix by increasing the width of the peritubular ring and in the early phases obliterating the tubule. Further calcification in the dentinal matrix, with the clumping together of some of the obliterated tubules and hypercalcified peritubular rings resulted in the zone of hypercalcified matrix. This pattern is similar to the original calcification of the dentinal and predentinal matrix, although exactly in reverse, since the most calcified zone is closer to the pulpal horn.

The evidence revealed in a careful perusal of the dental literature suggests that dentin is indeed a vital tissue, quite capable of reacting to certain stimuli. Dentin sclerosis is one observable change that has been documented both histologically and radiographically. A thorough consideration of its association with the carious process will be of value.

Television Measurement Instrumentation

Klein[47] in 1963 described a technique and instrumentation for intraoral clinical investigation, using the television microscope. Horwitz[48] in 1966 conducted a study to demonstrate the clinical application of the intraoral television microscope in measuring and assessing the buccal proximal marginal deterioration of the proximocclusal alloy restoration in deciduous teeth. The television intraoral microscope consisted of a petrographic microscope, coupled to both a television storage camera and a television camera. The margin image was viewed through the television camera. A switching control was triggered to release a shutter which activated the switching prism and took an electronically stored (Permachon) picture of the margin at 1/125 of a second.

Kerkhove[49] used the television densitometric instrumentation to radiographically evaluate the indirect pulp capping technique, using two base materials. The Benkow technique was utilized to facilitate repeated identical radiographs. These serial radiographs were positioned in an optoliner for purposes of constant illumination over the entire film. A television camera monitored the serial radiograph, with a step wedge, and presented this image on a television screen for densitometric readout. Thus, film densities of residual dentin beneath the two base materials could be recorded.

de Aguiar[50] utilized the television subtraction readout apparatus to count bone trabeculae and medullary spaces within a standardized sample area, from clinical radiographs of edentulous patients. This system allowed the operator to control electronically the density contrast, brightness and image reversal of the original radiographic information. The television optoliners were used as a duplicate constant light and optical system. One of each of the duplicate radiographs was placed within each optoliner and viewed by both television cameras. The television cameras were adjusted for super-imposition of both images, so that one camera carried a normal or positive image of one of the duplicate radiographs, while the other camera carried a reverse or negative image of the other duplicate radiograph. Therefore, the investigator could control radiographic shadows of overlying or underlying anatomic structures and so enhance the radiographic characteristics of the area of study.

STATEMENT OF PROBLEM

The purpose of this study was to evaluate the association between the depth of a carious lesion and the dentinal sclerosis produced beneath a calcium hydroxide methyl cellulose base material in a tooth restored with a silver amalgam alloy. The dimensional change in pulpal floor width and relative calcification of the produced sclerotic dentin overlying the pulp will be measured on the one, three, six and nine month postoperative serial radiographs, utilizing the television linear and densitometric instrumentation.

EXPERIMENTAL PROCEDURES

The experimental procedures section of this thesis has been divided into four parts, as follows:

1. Clinical operative procedures: The criteria for selection of teeth, cavity preparation, restorative materials utilized, and restoration placement,
2. Serial radiographic procedures: The technique for obtaining the initial and subsequent periodic, oriented serial radiographs,
3. Television instrumentation: The densitometric and linear measurement instrumentation, and
4. Measurement procedures: The procedures used in acquiring density and linear values from the serial postoperative radiographs.

Clinical Operative Procedures

The teeth selected for this study were from children in the mixed dentition stage selected from patients receiving treatment in the Pedodontic Department at Indiana University School of Dentistry. The sample consisted of maxillary and mandibular first and second deciduous molars and mandibular first permanent molars which met the following clinical criteria:

a) Teeth with deep caries and possible exposure of the dental pulp, as evidenced by a critical examination of the periapical and bitewing radiograph. (Figure 1)

b) No history of painful pulpitis or degenerative pulps as evidenced by clinical and radiographic observation.

c) No evidence of periapical pathology on the diagnostic periapical radiograph.

d) Teeth which were not sensitive to percussion.

e) Teeth with sufficient clinical crown to permit isolation by the rubber dam during the treatment and restorative procedures.

Prior to the operative procedure, the initial serial radiograph was taken. The teeth were anesthetized, utilizing infiltration procedures in the maxilla and mandibular block anesthesia in the mandible. A solution of two percent Xylocaine HCL with 1:100,000 epinephrine was used. The tooth was then isolated with the rubber dam.

Cavity preparation, as described by McDonald,[51] was then carried out, using a number 700 taper fissure bur in the air turbine. Carious dentin was scrupulously removed with spoon excavators and a round bur at speeds of 6,000 to 10,000 rpm. The presence or absence of a macroscopic pulp exposure was noted after complete caries removal. If an exposure was detected, it was recorded and the necessary pulp therapy was initiated. These teeth were no longer followed radiographically in this study. If there was no evidence of pulp involvement, the preparation was dried with air from the air syringe, and the pulpal floor was painted with a water soluble barium sulphate* solution, applied with a small ball burnisher. (Figure 2)

The application of barium sulphate facilitated radiographic identification of the relationship between the pulp chamber and the pulpal floor of the cavity preparation. The rubber dam was removed, the preparation was isolated with cotton rolls to prohibit moisture contamination, and a second serial radiograph was taken. A new rubber dam was then placed, the barium sulphate was scrupulously washed from the cavity preparation with water and then dried with air from the air syringe. A paste of calcium hydroxide methyl cellulose base material consisting of chemically pure calcium hydroxide and one percent methyl

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* Micropaque Powder, Demancy & Co. Ltd., England.

cellulose was then placed on the pulpal floor in the form of a creamy mixture with a ball burnisher. (Figure 3) The calcium hydroxide methyl cellulose base was then carefully tamped dry with a cottom pledget. All of the excess material was removed from the margins and walls of the cavity preparation with an explorer. A zinc phosphate cement base was placed when deemed clinically necessary. Finally, the preparation was restored with fine cut alloy* in a 1:1 ratio with mercury employing a mechanical+ amalgamator. The patient was then reappointed for polishing of the restoration. Subsequent serial radiographs were taken at intervals of one, three, six, and nine months.

Serial Radiographic Procedures

Reports have been published in the dental literature describing techniques for obtaining serial radiographs. Benkow[52] in 1957 described an adjustable appliance used for identical radiography and sterioradiography consisting of a film holder, distance rod, and focusing rings. Hollender and Lantz[53] in 1963 reported on an apparatus for serial identical roentgenography of the lateral parts of the lower jaw of the dog. The reliability of the technique was confirmed by obtaining parallax measurements

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* Caulk's 20th Century fine cut alloy, L.D. Caulk Co. Milford, Del.

\+ Wig-L-Bug Amalgamator, Crescent Dental Mfg. Co., Chicago, Ill.

of serial radiographs taken on different occasions. Dalitz[54] in 1964 also described a serial radiographic instrumentation. The apparatus varied from those previously described in that any region of the mouth could be studied utilizing five custom-made attachments, each consisting of a film holder, a square tube to accommodate its appropriate constant distance rod and a section for locating the compound impression of the approximating dentition. However, it was necessary for the patient to support the attachment with the fingers of one hand, in order to insure stability.

The requirements of this investigation, demanded a sturdier serial radiographic device than those previously described. Exposure times, temperature and age of the developing solutions, and developing times were recognized as possible variables. Therefore, an aluminum step wedge was incorporated into the film holder as a density control to verify the quality of the serial radiographs. A film holder was constructed for each individual tooth in the study. Two types of holders were fabricated, for right and left quadrants, of both maxilla and mandible.

DESCRIPTION OF APPARATUS:

The serial radiographic apparatus consisted of: (Figures 4, 5, 6, 7.)

1. A square metal x-ray head cone* attached to a standard 60 Kilovolt, 10 milliampere x-ray machine to replace the conventional plastic cone. The cone was further modified by constructing a snug slot for securing the constant distance rod.
2. Two aluminum constant distance rods+ were fabricated, one each for the right and left sides. Each rod was 11 3/8" long and formed in two planes, in order to center the film holder in the x-ray beam. The end of the rod was precisely slotted to fit completely over the aluminum step wedge in the film holder.
3. A precise aluminum step wedge was machined from Type KACC-00A-270-6061T6 3/8" x 1" aluminum bar. Five steps were cut in two m.m. thickness increments, each step measuring 4 m.m. in width.
4. A custom made film holder was fabricated of several small acrylic plastic parts to provide: (1) a film holder chamber; (2) a step wedge housing and distance rod support assembly mounted 90 degrees to the film chamber. The film holder chamber was 34 mm. wide

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* XRM Mfg. Corp. of America, Great Neck, N. Y.

\+ Kaiser Aluminum of America

± Kaiser Aluminum of America

by 30 mm. long with a 1 mm. by 25 mm. slotted area provided for the film pack. The step wedge housing provided the slot into which the distance rod fitted and a working surface for the compound bite.* The sides were made of 3 mm. thickness material with one end machined out 1 mm. by 7 mm., this latter area providing a bearing surface for the distance rod.

During the fabrication of the holder, its counterpart constant distance rod was positioned in the assembly over the wedge and liquid acrylic was introduced so that a precise fit, position and increased strength was achieved between the distance rod and the film holder.

Upon completion of assembly all square edges were polished to a smooth curved finish, and the lower film chamber area was rounded to permit easier fitting within the intraoral area.

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* Kerr's compound, Kerr Manufacturing Co., Detroit, Mich.

Television Densitometric and Linear Measurement Instrumentation

All electronic linear and density measurements were made in the Television and Electronic Dental Research Laboratory of Indiana University School of Dentistry (Figure 8). A schematic block diagram of the instrumentation is noted in Figure 9.

Steps two and four of the step wedge from each serial radiograph were calibrated as to density by the MacBeth Quanta Log Densitometer.* These particular steps were selected in order to calibrate the television digital instrumentation, as their density values were believed to be within the range of the control and sample area densities selected on each tooth studied. A constant flat illumination for the serial radiographs placed for television camera observation was provided by the television optoliners.+ The optical images from the television optoliners were converted to electrical signals in the television cameras. The images were presented to the video mixer-switcher for further processing.

<u>Television Densitometric Instrumentation</u>: The system utilized was that described by Klein and MacPherson.[55]

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* MacBeth Quanta Log, MacBeth Inst. Corp., Newburgh, N. Y.

+ TVO 1000 Optoliners, Photo Research Corp., Hollywood, Calif.

‡ VCF-3 Cameras, Sarkes Tarzian, Inc., Bloomington, Indiana

The operator had the opportunity to select any line of scan in the picture for density or linear measurement. An illuminated marker dot served as a density sampling probe and identified the area of measurement. The oscilloscope, employed to generate the density sampling probe signal was also used to display an enlarged portion of this selected scan line. This enabled the operator to see finite changes of density, depicted by variation in wave form. This verified that the sampling was being taken at the desired location of change.

The density measurement unit required that the television system must be first calibrated to a specific density scale.[56] The Quanta Log density measurements, as previously discussed, were used to derive this required density scale. The density sampling probe measured the video signal at the point selected in the radiograph, and provided an output signal to the digital readout. This reading was a function of density change of the area of sampling.

Linear Measurement Instrumentation: The line selector and oscilloscope equipment were identical to that of the density system. The marker dot position signal was sent to the linear unit, where offset and calibration controls were provided, therefore permitting the position of the dot to be set at a particular location and zeroed. The dot was then moved from the zeroed position to the desired point for measurement. This provided a signal to the digital readout which was calibrated to give a measure of the linear distance traversed by the marker dot.

Digital Display: The system was composed of a readout switching control, the digital readout display unit, and its associated television camera. The readout switching control enabled the operator to easily transfer from a density to a linear measurement. The digital readout unit, a digital voltmeter, provided instantaneous readings of selected measurements as microvoltage changes, of either density or linear change. This display viewed by its television camera, was electronically inserted into the upper portion of the master monitor image.

Video Image Mixing and Display: Klein and MacPherson[55] previously described this instrumentation. Four signal inputs originated from the two television optoliner camera systems, and two line selector spot positioning systems. The output from this mixer consisted of the density image, linear measurement image (which were presented to their individual oscilloscopes) and a composite radiographic image. A composite radiographic image, and digital readout image were presented to the special effects generator, where the digital readout information was inserted into the composite radiographic image. The output of this unit then was displayed on the master monitor at (14X) magnification for visual readout by the operator.

Measurement Procedures

Density Measurement: (Figure 10.) The television instrumentation was calibrated from the values obtained on steps 2 and 4 of each serial radiograph on the MacBeth Quanta Log Densitometer. The density sampling probe was then positioned on the area of study and a reading was obtained, while observing its wave form from the oscilloscope in a (5X) mode in order to exactly determine the area of density change. Subsequently, a second reading was obtained, in a similar manner, in the control area. The control area selected was on the opposite side of the same tooth, in an area of the same thickness of tooth structure, as the area of study. Both readings were recorded and the percent change in density (calcification) between the readings was calculated. This density measurement procedure was repeated for each serial radiograph.

Calculations: The television densitometric readings were based upon the calibrations made using the MacBeth Quanta Log Densitometer. In view of the range of densities in the radiographs, it was necessary to correct the readings to minimize error. A graph of a linear density scale was constructed on a logarithmic chart plotting opacity against transmission using the density-opacity-transmission table.[57] This table is based on the relationship that density equals

Log 10 opacity and transmission equals $\frac{1}{\text{opacity}}$. A sliding scale was constructed which matched the density scale, and permitted the operator to convert the density readings. This conversion was based on the averages calculated for steps 2 and 4 on the step wedge. These average densities were 1.12 for step 2 and 1.50 for step 4. (Figure 11) Upon conversion, the values were read as transmission. Therefore, all the density data was compared, within the same converted range, to derive a percent change in calcification.

Linear Measurement: (Figure 12.) The linear measurement unit was calibrated by a film type millimeter scale inserted in the television optoliner. The entire image of the scale, as well as small increments, were calibrated in order to insure maximum television camera linearity. Linear measurement on each serial radiograph was accomplished by rotating the radiographs in the television optoliner to such an angle, so as to place the bifurcation and selected measurement point on a horizontal scan line. The marker probe was positioned on the bifurcation point and the digital display adjusted to a zero readout. The measurement probe was then advanced to the point of measurement and the digital readout recorded. The linear measurement in the first operative serial radiograph was made from the bifurcation to that point on the identified radiopaque barium sulphate floor of the cavity which most closely approximated the pulp. A second measurement on this film was made from the bifurcation to the most extreme border of the pulp in the same plane. Both measurements were determined by observing finite density changes of the associated wave form display on the (5X) mode oscilloscope. The difference in these two readings represented a measurement of the original pulpal floor width. The procedure for the remaining one, three, six, and nine month radiographs was identical to that described for measuring to the pulp extremity. Differences in these measurements represented changes in pulpal floor width.

DATA

The purpose of this study was to determine whether there was a relationship between postoperative pulpal floor width dimensional change and the calcification change in the overlying pulpal dentin in deep cavities restored utilizing calcium hydroxide methyl cellulose base material. The sample of 44 teeth was divided into two groups, based on the thickness of the original pulpal floor prior to placement of the restoration.(Table 1) A pulpal floor width of 830 microns or less was arbitrarily selected as representing a very deep cavity based on clinical and radiographic interpretation. Twenty-four teeth satisfied this criteria and were placed in Group I. The remaining 20 teeth with pulpal floor widths ranging from 860 to 2370 microns were considered clinically deep cavities and placed in Group II.

Figures 13 and 14 demonstrate a graph for Groups I and II, which plots the average increase of pulpal floor width and percent change in calcification of the overlying pulpal dentin relative to postoperative time.

The average pulpal floor width in Group I (Figure 13) increased rapidly from 560 microns at the pre-restorative stage to 670 microns at one month, 810 microns at three months, 880 microns at six months and 980 microns at nine months. The percent change in calcification at one month was 37 percent, which then decreased dramatically to 20 percent at three months, and increased again to 32 percent at six months. At nine months, the percent calcification again decreased to 20 percent.

The average pulpal floor width for Group II (Figure 14) increased steadily. At the pre-restorative stage, the pulpal floor measured 1410 microns, 1460 microns at one month, 1610 microns at three months, 1750 microns at six months, and 1830 microns at nine months. The percent change in calcification at one month was 29 percent. A slight decrease to 26 percent occurred at three months. Again, as in Group I, the calcification showed an increase, reaching 35 percent at the six month period and remained at 35 percent through nine months.

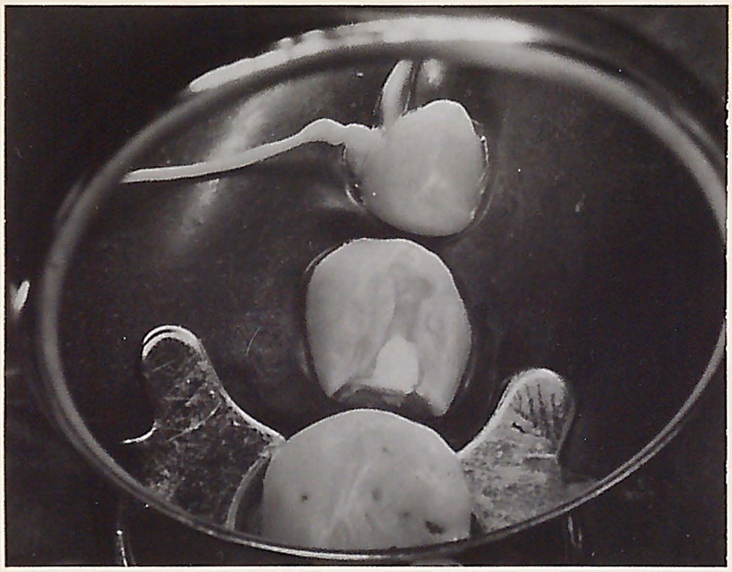
A bar graph (Figure 15) demonstrates the rate of change as a percent change in pulpal floor width for Groups I and II. In Group I the percent change at one month was 19.9 percent, in contrast to a 3.5 percent increase in Group II for the same period. At three months, the percent change for Group I was 44.6 percent, while Group II indicated a change of only 13.4 percent. This pattern continued again with Group I demonstrating a rapid increase of 57.1 percent change in pulpal floor width at six months and 73.9 percent at nine months. Comparatively, Group II showed a minimum percent change increase in pulpal floor width of 24 percent at six months, and 29.7 percent at nine months. A second bar graph (Figure 16) compares the percent change in calcification for Groups I and II. In Group I, the percent change in calcification at one month was 37 percent, in contrast to a 29 percent increase in Group II for the same period. At three months, the percent changes in calcification for Groups I and II decreased to 20 percent and 26 percent respectively. At the six month period, Group I again increased to 32 percent, and decreased to 20 percent at nine months. Group II also increased to 35 percent at six months remaining constant at 35 percent through the nine month period.

ILLUSTRATIONS AND TABLES

Figure 1. A preoperative radiograph demonstrating the extent of carious involvement of a first deciduous molar selected for this study.

Figure 2. The water soluble barium sulphate applied to the pulpal floor of the cavity preparation after complete removal of carious dentin which will identify the initial thickness of the overlying pulpal dentin.

Figure 3. The placement of the calcium hydroxide methyl cellulose base material prior to the amalgam restoration.



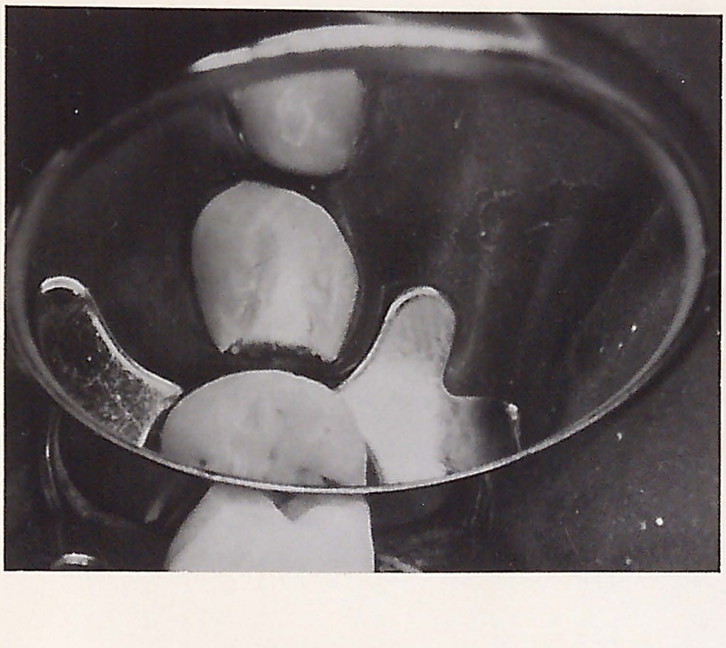


Figure 4. The instrumentation used for obtaining the serial radiographs.

A. The right constant distance rod.

B. The metal cone which is attached to the head of the x-ray machine.

C. Custom made acrylic film holder.

D. Film holder with compound bite

Figure 5. The clinical application of the serial radiographic instrumentation.

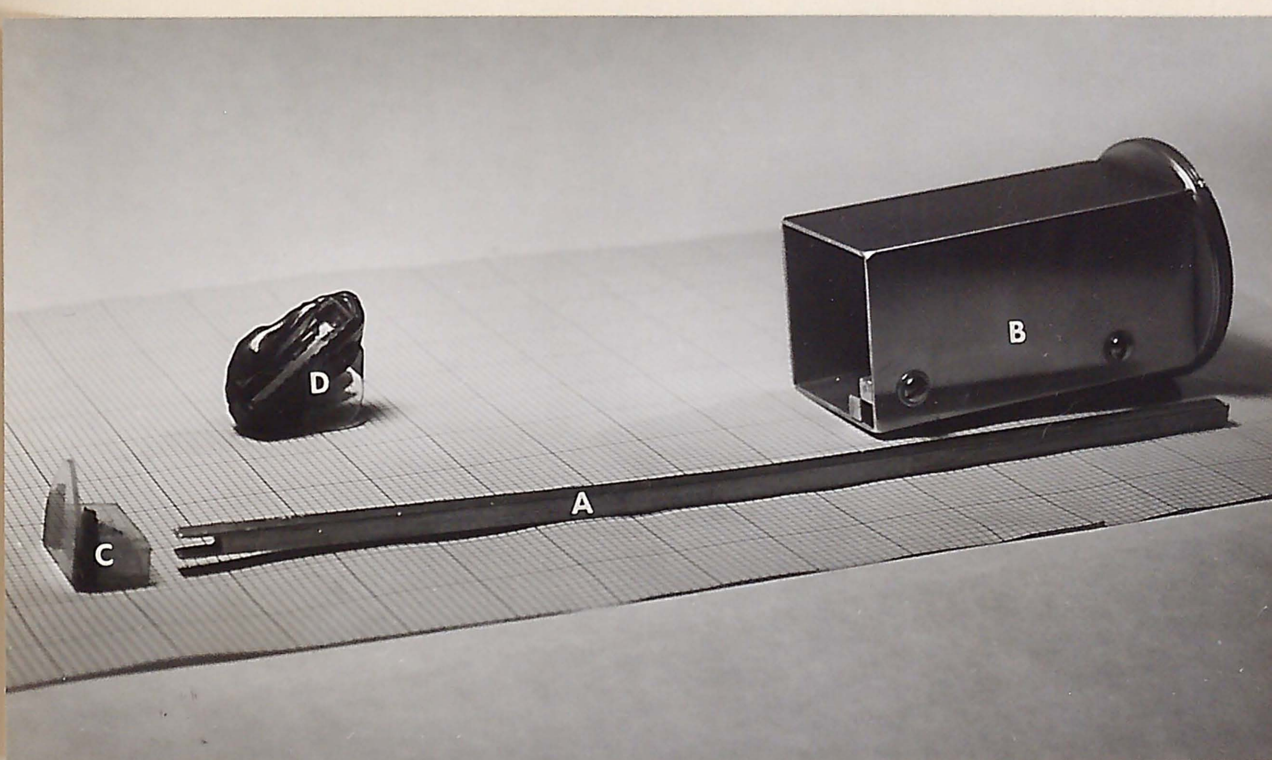
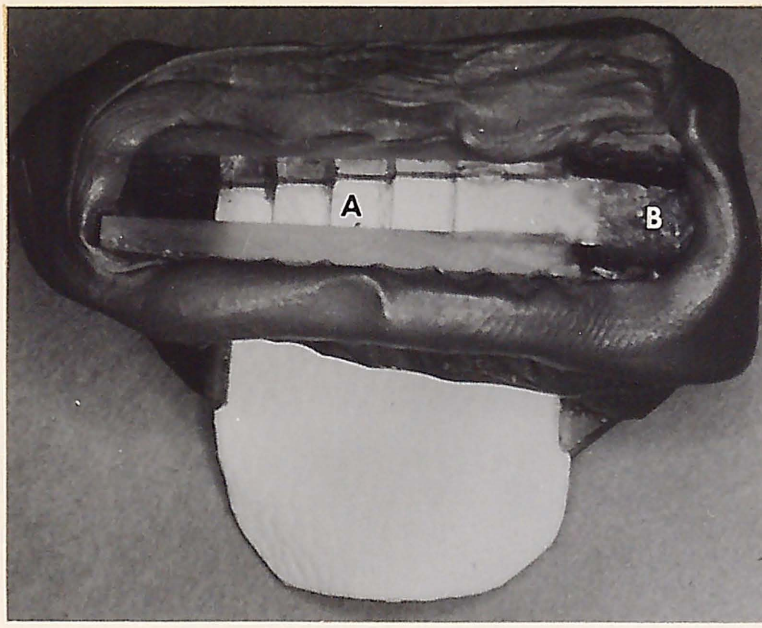

D
B
A
C

Figure 6. The custom-made acrylic film holder with its aluminum step wedge (A) and slot (B) for the insertion of the constant distance rods.

Figure 7. Posterior view of the film holder, depicting the compound bite index and housing for the Type One film pack.


A
B

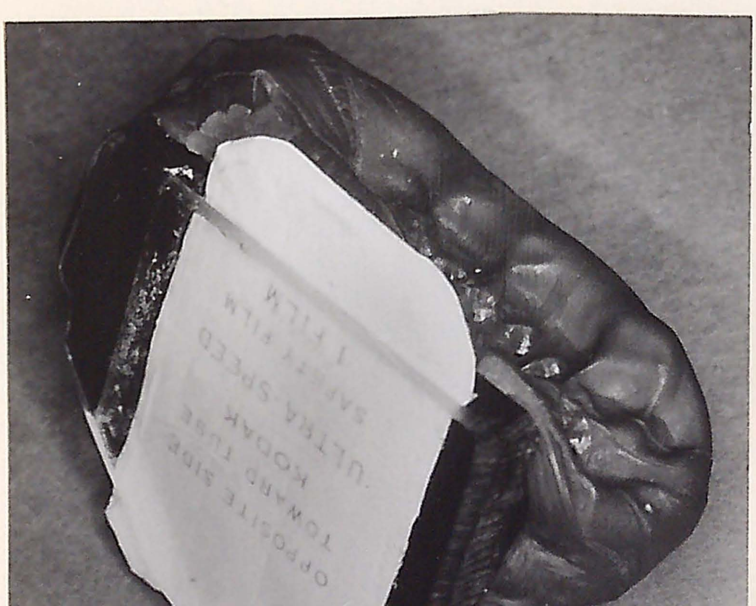

OPPOSITE SIDE
TOWARD TUBE
KODAK
ULTRA-SPEED
SAFETY FILM
1 FILM

Figure 8. An overall view of the laboratory and instrumentation used for density and linear measurements.

A. MacBeth Quanta Log

B. Television optoliners and associated television cameras.

C. Line selector oscilloscope

D. Digital display system and associated television camera

E. Master monitor

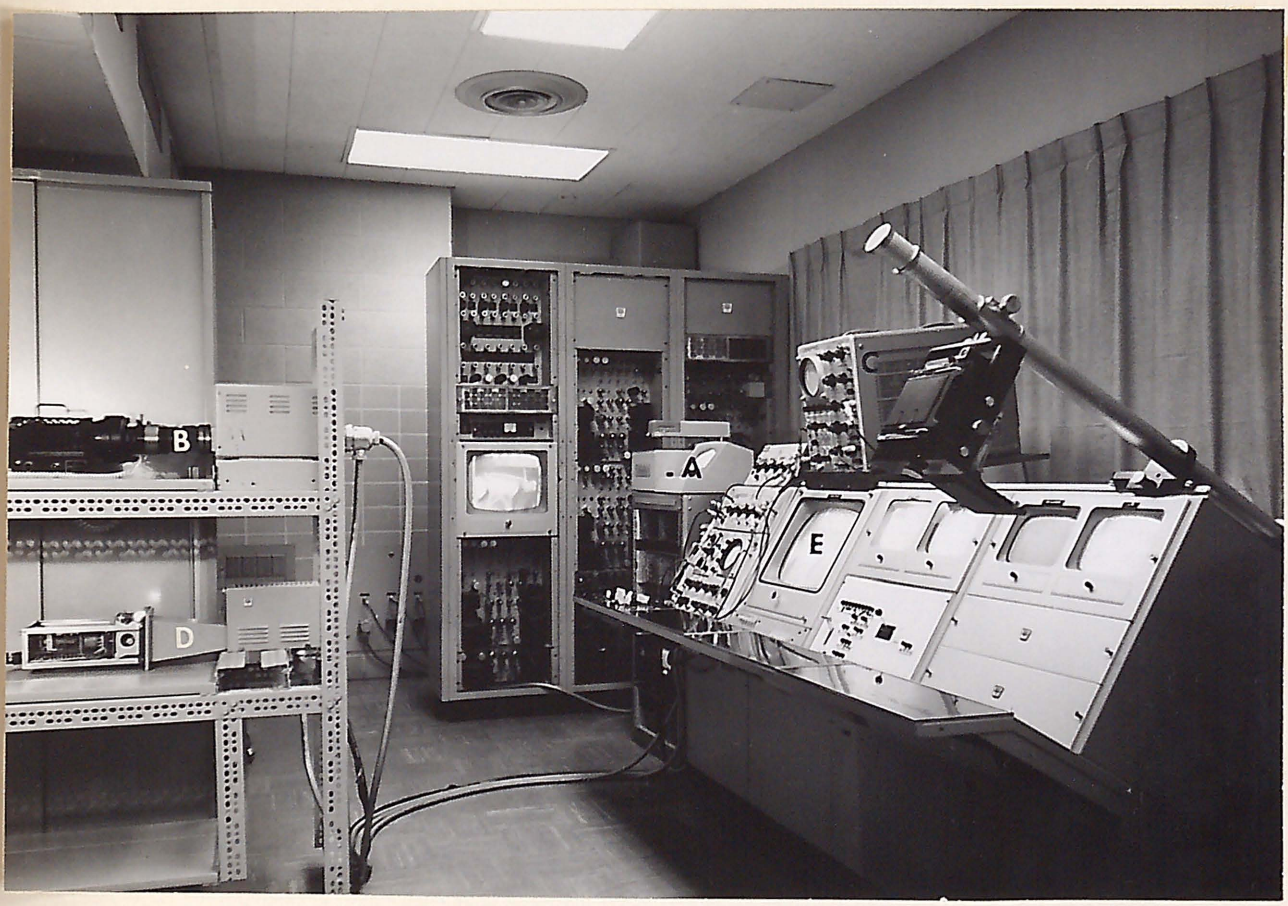

B
D
A
E

Figure 9. Television linear and densitometric instrumentation. This schematic block diagram illustrates the application and basic components of the system used for obtaining density and linear measurements. The barium sulphate film was placed in the number one optoliner, and the serial radiographs were placed in the number two optoliner. Each image is viewed on its respective monitor. The barium sulphate film identified the distance between the bifurcation and the deepest point on the pulpal floor. The distance between the bifurcation and the pulp horn extremity was measured from the one, three, six, and nine month serial radiographs in optoliner two. These images were presented to the video-mixer switcher, and in turn, to the oscilloscope density and linear measurement units. These units allowed the selection of that portion of the scan line upon which the measurements were to be made. The digital display system presented either linear or density measurements. This display and the mixed images were combined in the special effects generator where the display was inserted into this image. The combined image, consisting of the radiograph selected scan lines, for both density and linear measurement and the digital display system were presented on the master monitor for investigator viewing.

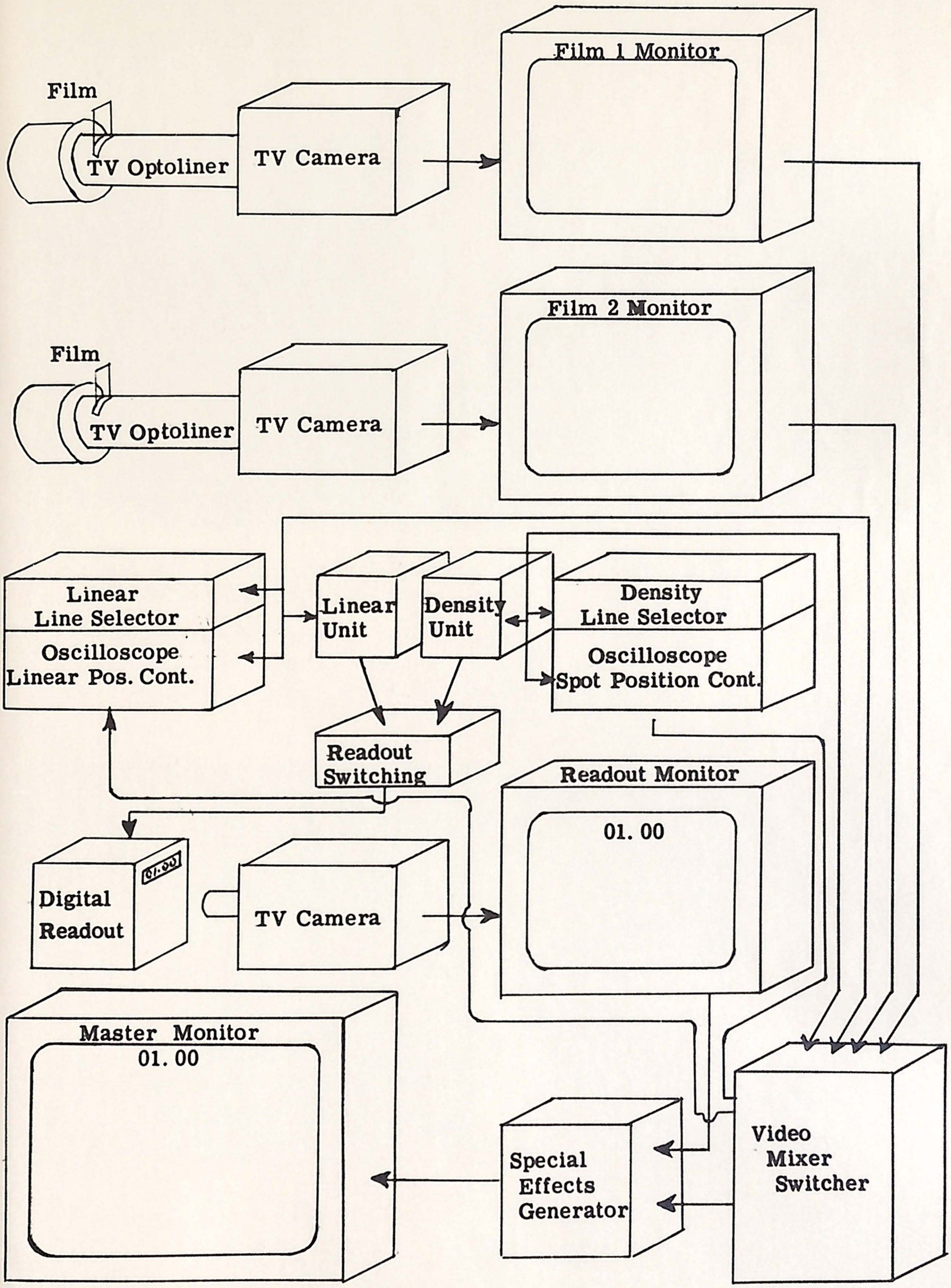
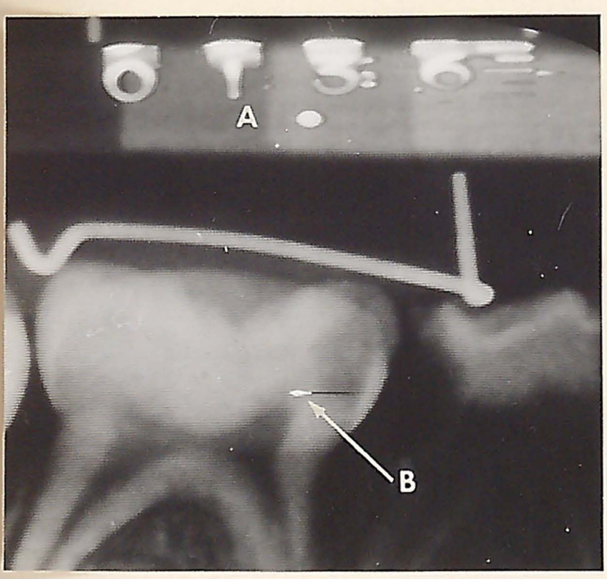

Film
TV Optoliner
TV Camera
Film 1 Monitor
Film
TV Optoliner
TV Camera
Film 2 Monitor
Linear Line Selector
Oscilloscope Linear Pos. Cont.
Linear Unit
Density Unit
Density Line Selector
Oscilloscope Spot Position Cont.
Readout Switching
Readout Monitor
01. 00
Digital Readout
01.00
TV Camera
Master Monitor
01. 00
Special Effects Generator
Video Mixer Switcher

Figure 10. (a) Preoperative radiograph of a deep carious lesion illustrating the density step wedge (A) and measurement marker probe (B) with density value readout on the digital display.

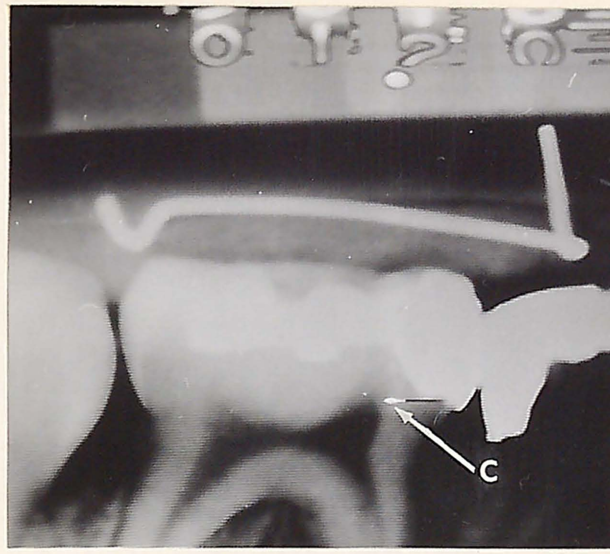
(b) Three month radiograph depicting "A white area" (C) of sclerosis with its increased opacity.

(c) Six month radiograph with a further increase in calcification (D).

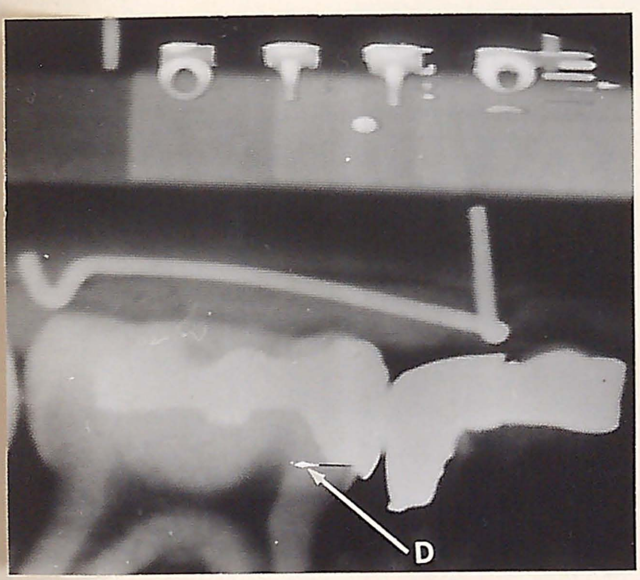
(d) Nine month radiograph of the same series with same area of dentin sclerosis at (E).



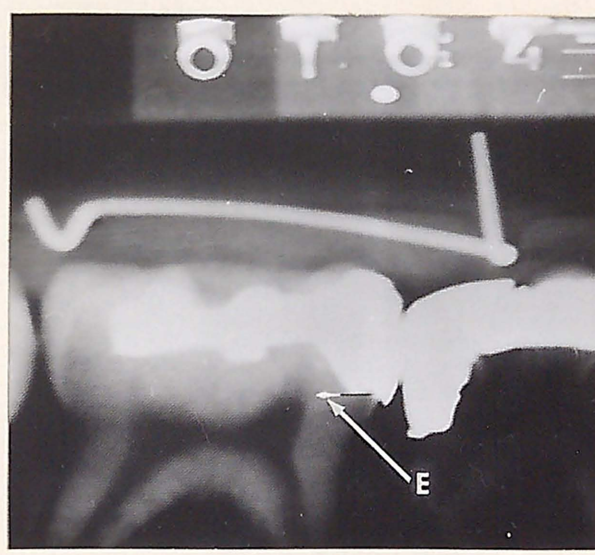
a



b

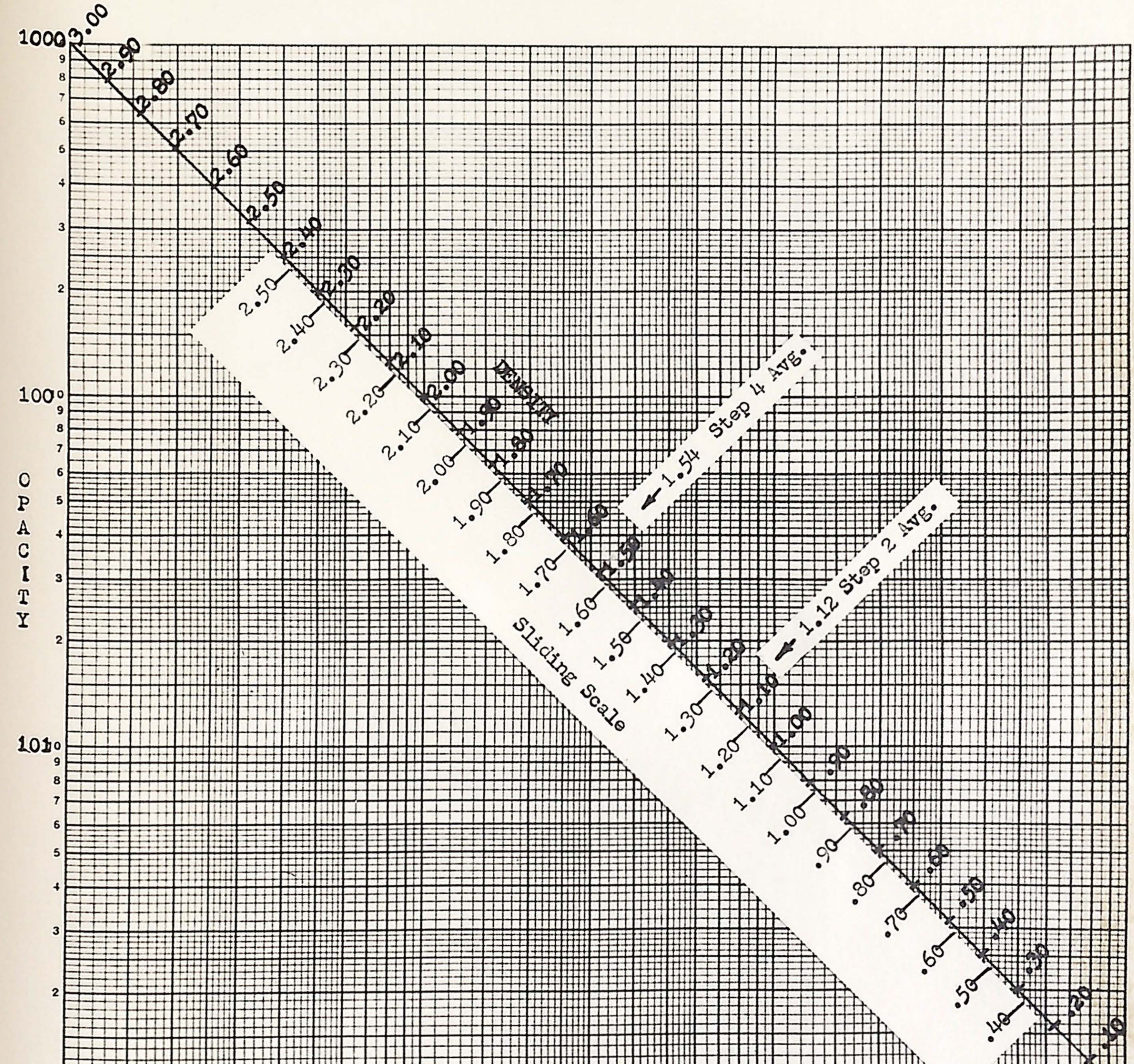


c



d

Figure 11. The density conversion sliding scale, using the density-opacity-transmission table. This permitted conversion of the density values which were based on the average densities for steps 2 and 4 on the step wedge. Therefore, it was possible to make all density comparisons within the same density range.



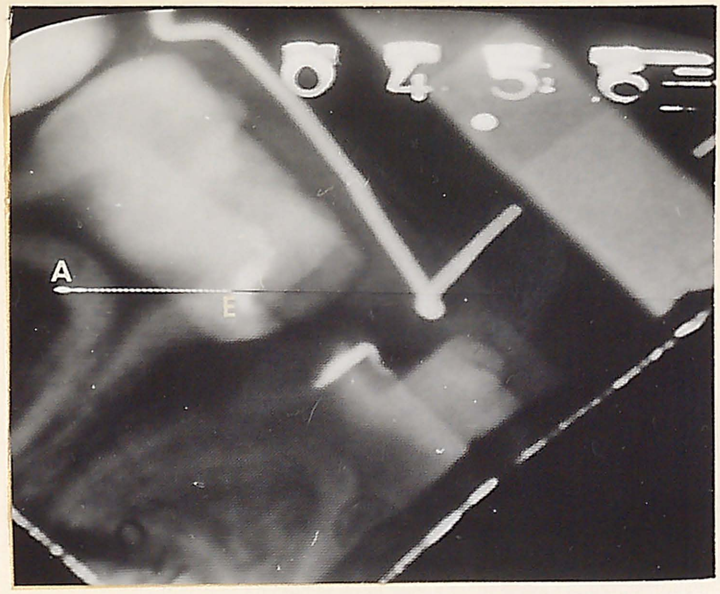
OPACITY
TRANSMISSION
0.1
1.0
10
100
1000
100
10
1.00
DENSITY
Sliding Scale
1.54 Step 4 Avg.
1.12 Step 2 Avg.

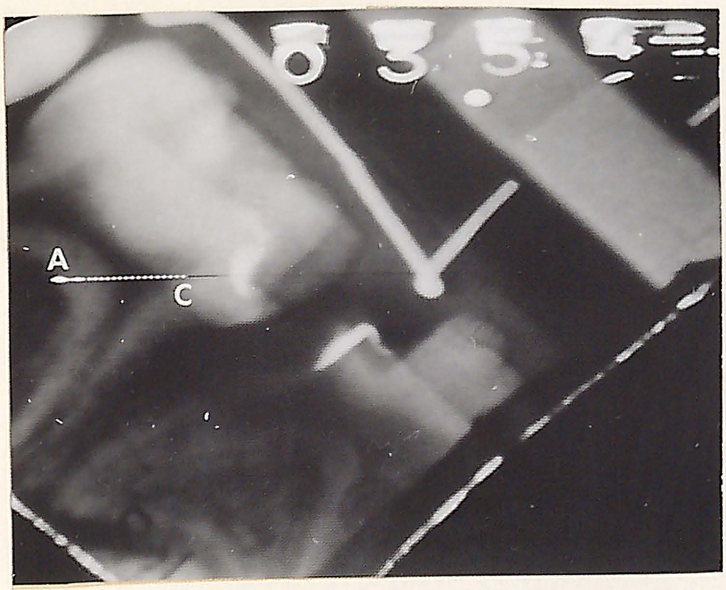
Figure 12. Master monitor photographs demonstrating the linear measurement procedure.

(a) The pre-restorative radiograph: Distance between bifurcation (A) and barium sulphate (E) as depicted by the image of the measurement marker probe. Digital display system reading 4560 microns as distance between (A) and (E).

(b) The same radiograph on an identical horizontal plane with the digital display system reading 3540 microns as the distance between the bifurcation (A) and the pulp horn extremity (C).

(c) Three month serial radiograph showing a decreased linear measurement of 3440 microns (A) from the bifurcation to the pulp horn extremity (C).


A
E


A
C

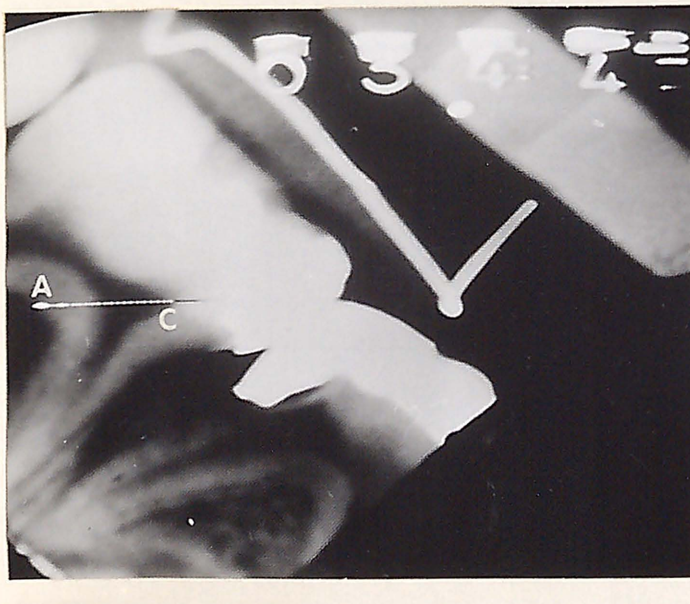
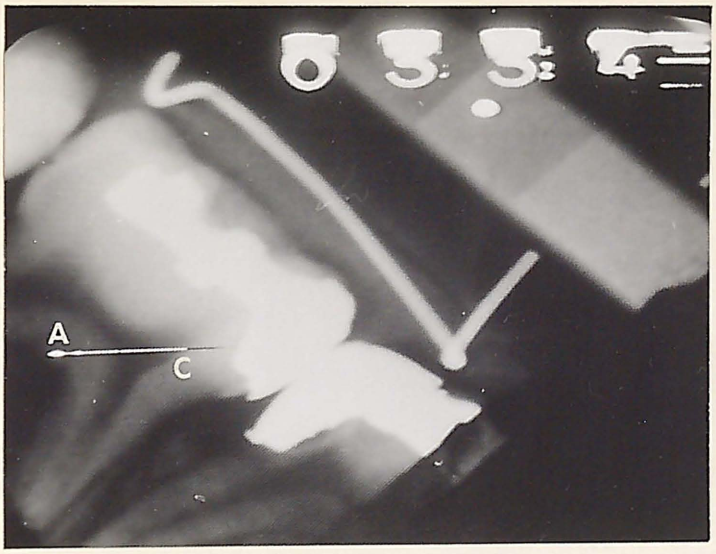

A
C

Figure 12. (continued)

(d) Six month radiograph with further linear decrease to 3340 microns of the bifurcation (A) to pulp horn (C) distance.

(e) Nine month radiograph with another decrease in the bifurcation (A) to pulp horn (C) distance, now measuring 3250 microns.


A
C

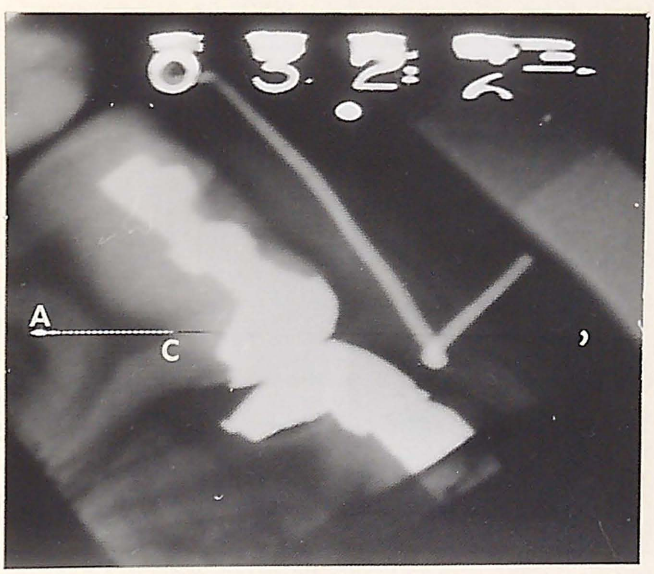

A
C

Table 1. The relationship of the pre-restored pulpal floor width to the postoperative pulpal floor average change in width and percent change in calcification.

No. Teeth - division of teeth into two groups. Group I - clinically very deep cavities. Group II - clinically deep cavities.

Pulpal Floor Width Range (microns) - the range of widths in microns of the pulpal floor after all carious dentin was removed.

Average Pulpal Floor Width (microns) - the average postoperative dimensional changes, relative to time in pulpal floor width.

Average Percent Change in Calcification - the average percent change, relative to time of the overlying pulpal dentin.

	NO. TEETH	PULPAL FLOOR WIDTH RANGE (MICRONS)	AVG. PULPAL FLOOR WIDTH (MICRONS)					AVG. % CHANGE CALCIFICATION			
			Pre-Rest	1 mos.	3 mos.	6 mos.	9 mos.	1 mos.	3 mos.	6 mos.	9 mos.
GROUP I	24	200-830	560	670	810	880	980	37	20	32	20
GROUP II	20	860-2370	1410	1460	1610	1750	1830	29	26	35	35

Figure 13. The Group I average pulpal floor width in microns and calcification change in percent.

AVERAGE PULPAL FLOOR WIDTH AND CALIFICATION CHANGE

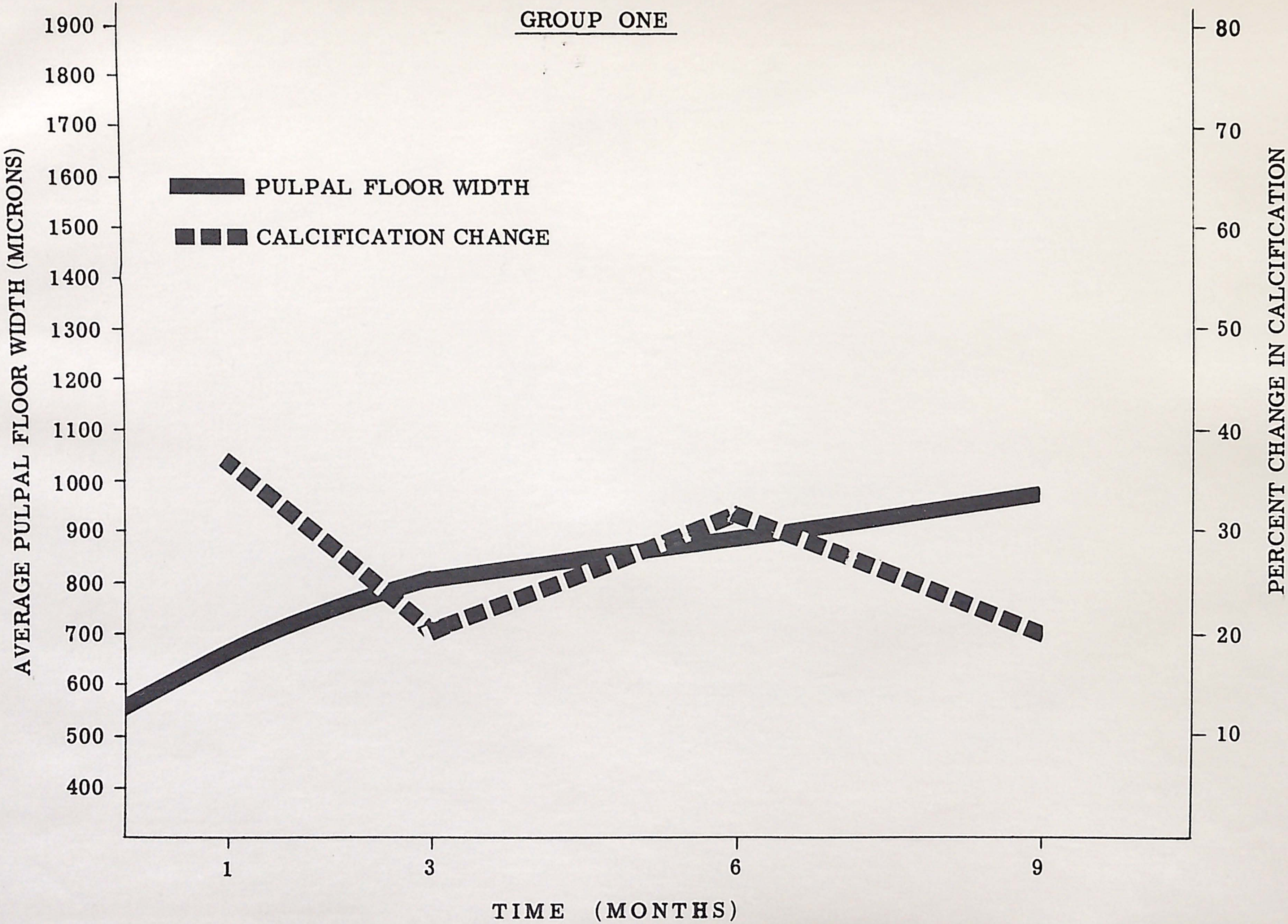


Figure 14. The Group II average pulpal floor width in microns and calcification change in percent. Group II indicated a similar pattern to Group I however a much slower response in dimensional change of the pulpal floor width and a less pronounced increase in calcification during the nine month period of study.

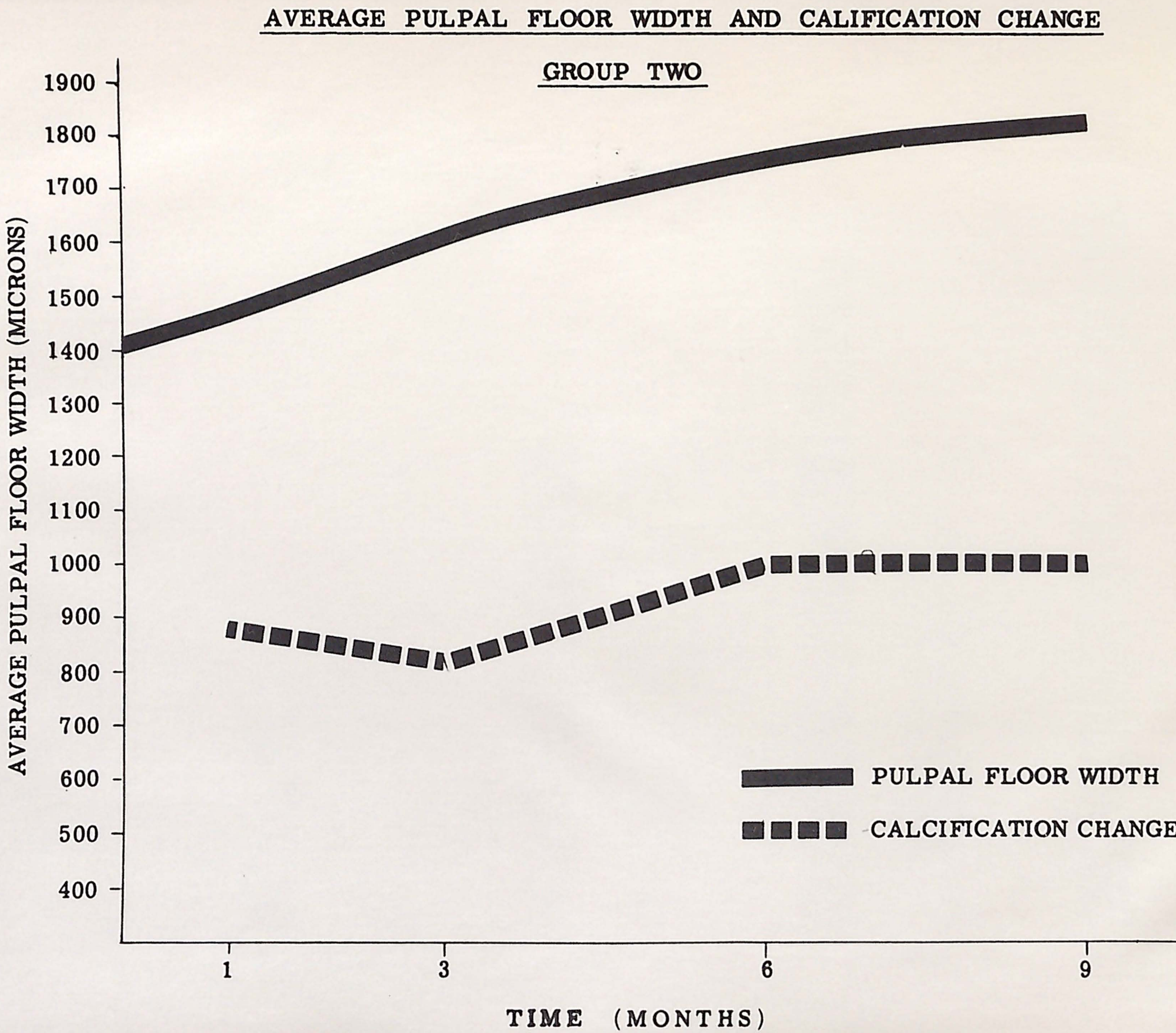
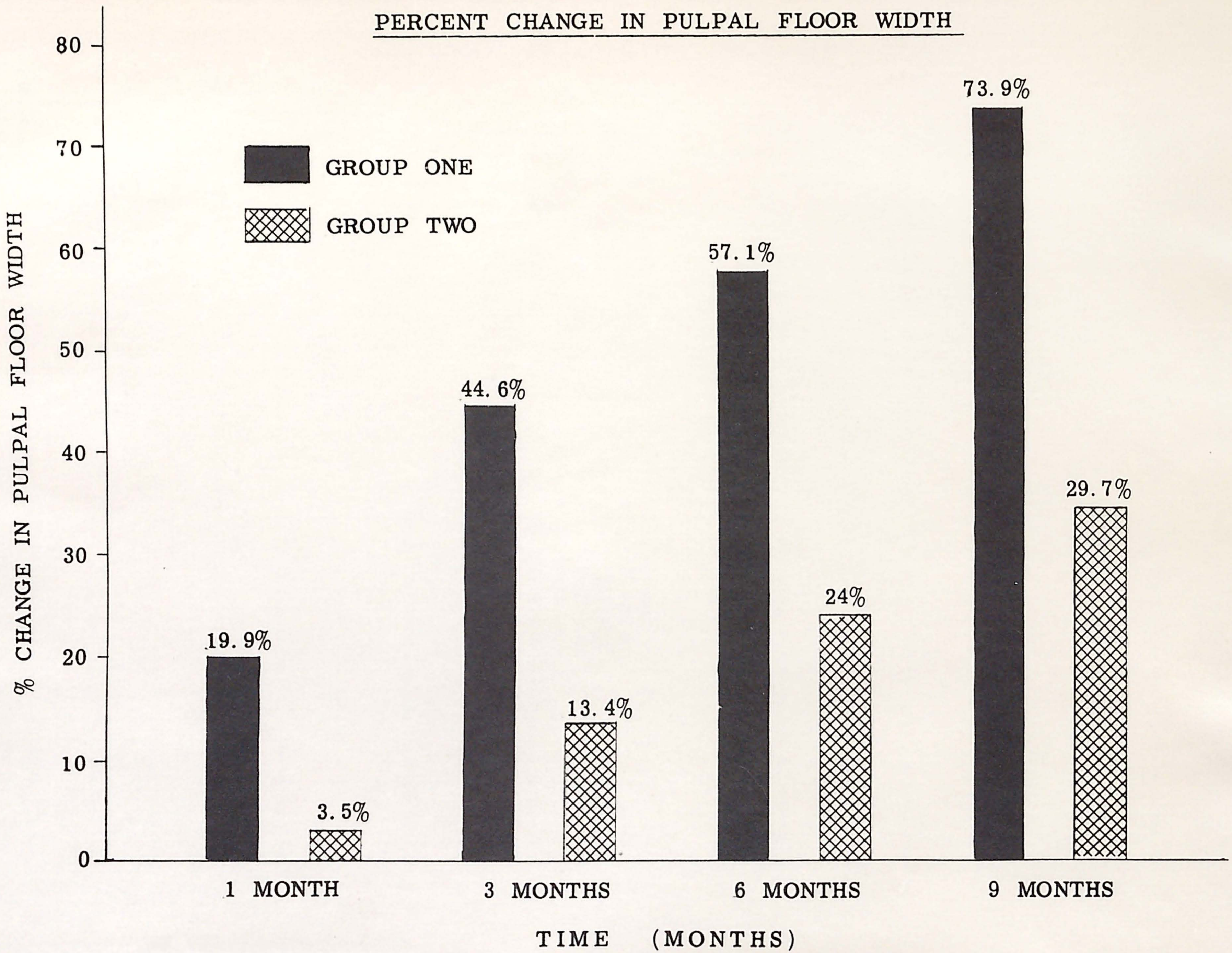

AVERAGE PULPAL FLOOR WIDTH AND CALIFICATION CHANGE
GROUP TWO
AVERAGE PULPAL FLOOR WIDTH (MICRONS)
1900
1800
1700
1600
1500
1400
1300
1200
1100
1000
900
800
700
600
500
400
PERCENT CHANGE IN CALCIFICATION
80
70
60
50
40
30
20
10
1
3
6
9
TIME (MONTHS)
PULPAL FLOOR WIDTH
CALCIFICATION CHANGE

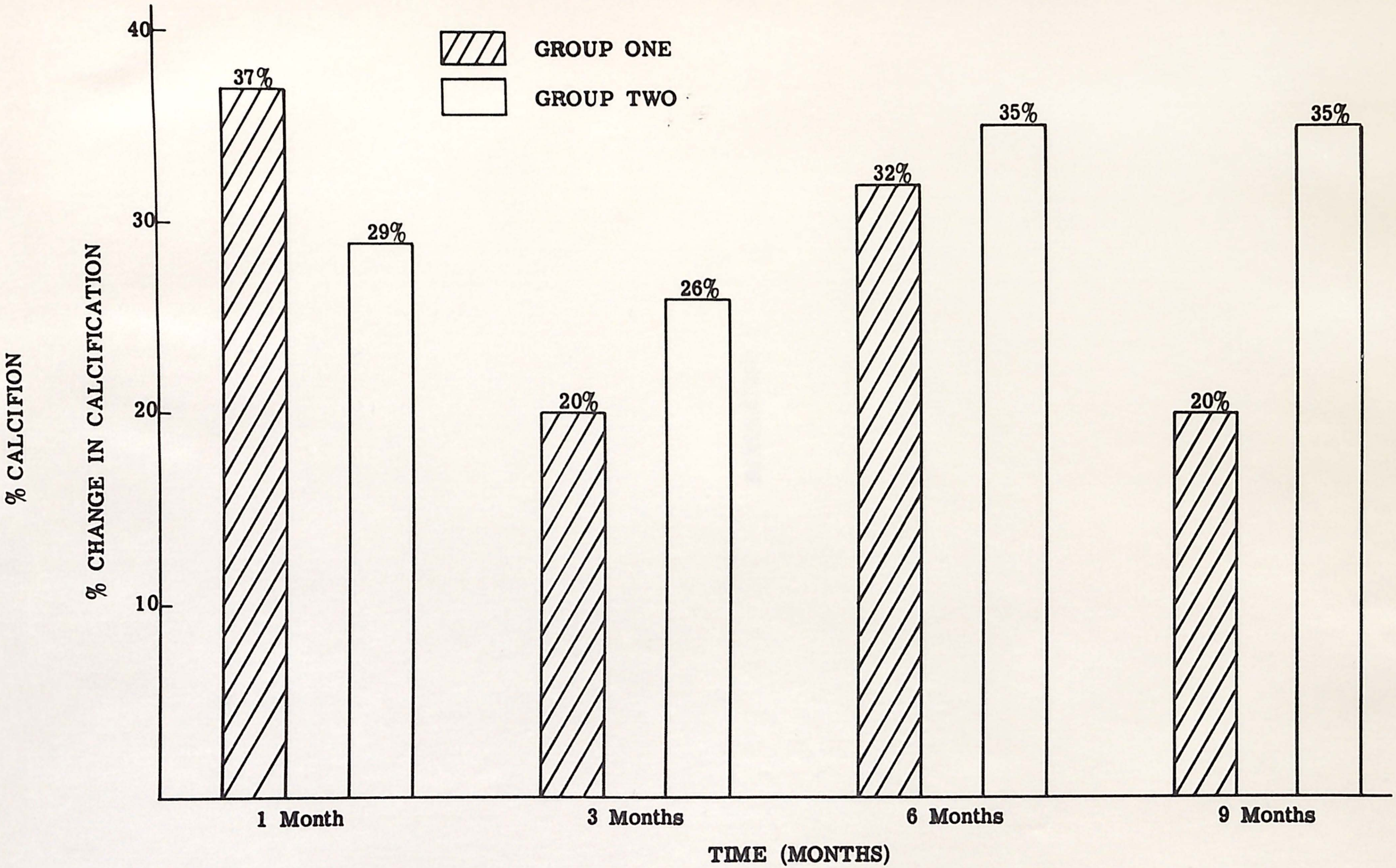
Figure 15. A comparison of the percent rate of change in pulpal floor width for Groups I and II. The thinner the pre-restored pulpal floor (Group I), the more rapid and dramatic is the postoperative pulpal floor increase in width, which is apparently a protective pulpal response.



PERCENT CHANGE IN PULPAL FLOOR WIDTH
80
70
60
50
40
30
20
10
0
% CHANGE IN PULPAL FLOOR WIDTH
GROUP ONE
GROUP TWO
19.9%
3.5%
44.6%
13.4%
57.1%
24%
73.9%
29.7%
1 MONTH
3 MONTHS
6 MONTHS
9 MONTHS
TIME (MONTHS)

Figure 16. A comparison of the percent change in calcification for Groups I and II. During the periods of time when a significant increase in pulpal floor width was noted, the percent change in calcification decreased. Conversely, when little pulpal floor increase occurred, the percent change in calcification increased.

PERCENT CHANGE IN CALCIFICATION



DISCUSSION

The objective of this study was to evaluate the association between the depth of a carious lesion and the dentinal sclerosis produced beneath a calcium hydroxide methyl cellulose base material.

The data indicates that those teeth in Group I, where initial pre-restorative pulpal floor widths ranged from 200 to 830 microns, clinically a very deep cavity, demonstrated a rapid and quantitative increase in pulpal floor width during the nine month period of study. Initially, the percent increase in calcification of the overlying pulpal floor demonstrated its highest peak of calcification at the one month postoperative period, apparently the calcification occurred as a protective pulpal response, through a very thin overlying layer of dentin. As the initial overlying layer of dentin increased in width, the greater its increase, the less the calcification change. Therefore, the increase in calcification is apparently a function of the width of the overlying pulpal dentin in those teeth with initially very deep cavities (Group I). Those teeth in Group II whose pulpal floor widths ranged from 860 to 2370 microns, clinically a deep cavity, indicated a similar pattern to the teeth in Group I. The relationship between pulpal floor width and calcification change again was a function of the initial pre-restorative pulpal floor width. These teeth, with a clinically deep cavity and a greater initial pulpal floor width, demonstrated that during the periods of time when a significant increase in pulpal floor width was noted, the percent changes in calcification decreased. Conversely, when little pulpal floor width occurred, the percent change in calcification increased. No differences were noted between deciduous and permanent teeth.

One of the criteria for the selection of teeth for this study was that these teeth should radiographically indicate the possibility of a pulp exposure. Initially, of the 27 teeth selected for Group I, clinically the very deep carious lesion, it is interesting that in only three instances were carious exposures encountered.

The following factors could have introduced some error into the study. Many of the children were in the mixed dentition stage at the time of onset of this study. Therefore, difficulty was occasionally encountered in replacing the compound bite index and its associated film holder into the mouth with the assurance that the relationship of teeth to the radiograph remained constant. The developing of the radiographs was not critically controlled. The imbedded step wedge, and MacBeth Quanta Log were employed to compensate for this, but nevertheless, it was impossible to eliminate this error completely. Kerkhove,[49] in conducting a densitometric study, stated that it was very difficult to orient and locate the same area that was being measured on each radiograph. However, in this study, the oscilloscope was employed to generate the density sampling probe signal, and display an enlarged portion of the selected scan line. This enabled the operator to see finite changes of density and verified that the sampling was being taken at the desired location on each serial radiograph.

The importance of this study lies in the fact that calcium hydroxide methyl cellulose indeed acted as a "trigger mechanism" initiating dentin to react to a specific stimulus by depositing sclerotic dentin. The extent to which this reaction occurred depended upon the initial width of the pulpal floor and its associated dimensional and calcification changes with time.

SUMMARY AND CONCLUSIONS

The purpose of this study was to evaluate the association between the depth of a carious lesion and the dentinal sclerosis produced beneath a calcium hydroxide methyl cellulose base material. The sample consisted of a total of 41 first and second deciduous molars and 3 first permanent molars from children in the mixed dentition stage that clinically demonstrated deep cavities with no pulpal involvement.

Prior to the operative procedure, the initial serial radiograph was taken using the custom-built acrylic film holder with imbedded density step wedge, in association with its counterpart constant distance rod. The tooth was then isolated with the rubber dam and cavity preparation was carried out, using a number 700 taper fissure bur in the air turbine. The carious dentin was scrupulously removed with spoon excavators and a round bur. The presence or absence of a macroscopic pulp exposure was noted. If there was no evidence of pulp involvement, the preparation was dried, and the pulpal floor was painted with a water soluble barium sulphate solution. A second serial radiograph was taken at this time. A new rubber dam was then placed, all traces of the barium sulphate were flushed from the cavity with water and a creamy mixture of calcium hydroxide methyl cellulose base material was placed on the cavity floor. The tooth was then restored with silver amalgam alloy. The patient was then reappointed for polishing of the restoration. Subsequent oriented serial radiographs were taken at intervals of one, three, six, and nine months.

The television instrumentation was calibrated for density measurements from the values obtained on steps 2 and 4 from the step wedge of the clinical radiographs, using the MacBeth Quanta Log. The density sampling probe was then positioned on the area of study and a reading was obtained, while observing the wave form for that particular area, on the oscilloscope. A second control reading was taken in an area of the same thickness of tooth structure, as the study area, on the opposite side of the same tooth. Both readings were recorded and the percent change in density (calcification) between the readings was calculated. Linear measurements on each serial radiograph were accomplished by rotating the radiographs in the television optoliner to such an angle so as to place the bifurcation and selected measurement point on a horizontal scan line. The digital display was adjusted to a zero readout, and the measurement probe was then advanced to the points of measurement for linear readout. The distance from the bifurcation to that point on the barium sulphate which most closely approximated the pulp minus the distance from the bifurcation to the pulp horn extremity represented the pre-restorative thickness of the pulpal floor. Both density and linear measurements of the pulpal floor were made for each subsequent serial radiograph.

The sample of 44 teeth was divided into two groups based on the thickness of the original pulpal floor prior to restoration placement. A width of 830 microns or less was arbitrarily selected as representing a very deep cavity, based on clinical and radiographic interpretation. Twenty-four teeth satisfied this criteria and were categorized as Group I. The remaining 20 teeth were considered clinically deep cavities and were placed in Group II.

The data indicate that those teeth in Group I whose initial preoperative pulpal floor width ranged from 200 to 830 microns, clinically a very deep cavity, demonstrated a rapid and quantitative increase in pulpal floor width during the nine month period of study. Initially, the percent increase in calcification of the overlying pulpal floor demonstrated its highest peak of calcification at the one month postoperative period, apparently, a protective pulpal response, through a very thin layer of dentin. Those teeth in Group II, clinically a deep cavity whose pulpal floor widths ranged from 860 to 2370 microns indicated a similar pattern, however a much slower response in dimensional change of the pulpal floor and a less pronounced increase in calcification than the teeth in Group I. The relationship between pulpal floor width and calcification change, again was a function of the initial pre-restorative pulpal floor width. No differences were noted between deciduous and permanent teeth.

Conclusions

1. A calcium hydroxide methyl cellulose base material acts as a "trigger mechanism," in deep cavities, for stimulating the deposition of sclerotic dentin. The rate at which this reaction occurs is dependent upon the initial width of the pulpal floor, prior to placement of the base material and the restoration.
2. The thinner the initial pre-restored pulpal floor, the more rapid and dramatic is the postoperative pulpal floor increase in width, which is apparently a protective pulpal response.
3. During the periods of time when a significant increase in pulpal floor width was noted, the percent change in calcification of this area decreased. Conversely, when little pulpal floor width increase occurred, the percent change in calcification increased.
4. The results of this study indicate that it is not possible to diagnose the presence of a pulp exposure by radiographic methods.
5. The television linear and densitometric instrumentation is a precise clinical research tool, which can identify and measure radiographic changes ordinarily not visible macroscopically.

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February 11, 1941	Born in Toronto, Ontario
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ABSTRACT

A TELEVISION RADIOGRAPHIC EVALUATION OF THE ASSOCIATION BETWEEN DENTIN SCLEROSIS AND PULPAL FLOOR WIDTH

by Julian Sheldon Geller, Toronto, Canada

The purpose of this investigation was to evaluate the association between the depth of a carious lesion and the sclerotic dentin deposited beneath a calcium hydroxide methyl cellulose base material. The sample chosen consisted of teeth with deep caries and possible pulp exposure, as evidenced by a critical radiographic examination. Clinical procedures consisted of a preoperative serial radiograph, followed by complete caries removal. A barium sulphate radiopaque solution was then applied to the base of the preparation, followed by a second serial radiograph. The barium sulphate was removed and a calcium hydroxide methyl cellulose base was applied and the tooth restored with a silver amalgam alloy. Subsequently one, three, six, and nine month serial radiographs were taken postoperatively. Calcification change of sclerotic dentin overlying the pulp was measured in relation to pulpal floor width by the television instrumentation.

The conclusions of this study are as follows:

1. A calcium hydroxide methyl cellulose base material acts as a "trigger mechanism," in deep cavities, stimulating the deposition of sclerotic dentin.
2. The thinner the initial pre-restored pulpal floor, the more rapid and dramatic is the post-operative pulpal floor increase in width, which is apparently a protective pulpal response.

3. During the periods of time when a significant increase in pulpal floor width was noted, the percent change in calcification of this area decreased. Conversely, when little pulpal floor width increase occurred, the percent change in calcification increased.